Original Article

Efficacy and Safety of Strict Voltage-based Substrate Mapping and Radiofrequency Catheter Ablation in Electrical Storms—Review of Substrate-mapping Guided Ablation in Frequent Appropriate Shocks

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Background: We investigated the efficacy and safety of strict voltage-based substrate mapping and radiofrequency catheter ablation (SV-substrate-map ablation) in patients with electrical storm.

Methods and Results: SV-substrate-map ablation was performed in 15 patients suffering from multiple appropriate shocks (6.0 ± 3.8/day) from implantable cardioverter defibrillators (ICDs). Strict voltage criteria were defined as: non-arrhythmogenic areas, >0.6 mV; low voltage areas, >0.1 to ≤ 0.6 mV; and scar, ≤ 0.1 mV. Using an electroanatomic mapping system, catheter ablation was performed at every possible arrhythmogenic region inside the low voltage areas. Further, we presented a review of the literature and investigated the published data on substrate-mapping guided ablation for electrical storm. After repeat endocardial ablation procedures in 4 patients and an epicardial approach in one, the targeted ventricular tachycardias (VTs) were successfully ablated, and the electrical storms were completely controlled in all. During a mean follow-up period of 801 ± 409 days, only one VT followed by an appropriate ICD shock was observed. No potential complications occurred during the procedure or follow-up. Our results were comparable or better than that of previous substrate-mapping guided ablation studies.

Conclusion: SV-substrate-mapping ablation may be effective and safe for resolving serious clinical situations and prolonging the longevity of ICD devices in patients with electrical storms.

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Key words: Substrate mapping, Electrical storm, Catheter ablation, Implantable cardioverter defibrillator, Epicardial

Introduction

Although implantable cardioverter defibrillators (ICDs) have become the main therapeutic tool for patients with life-threatening ventricular arrhythmias,[1–3] patients with an ICD often require con-

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comitant therapy with antiarrhythmic agents to decrease the frequency of defibrillator therapies.[4] Despite concomitant antiarrhythmic therapy, some patients still have frequent episodes of ventricular tachycardia (VT), resulting in numerous shocks or antitachycardia pacing therapies. In such a setting, radiofrequency (RF) catheter ablation may be effective for preventing frequent ICD deliveries of electrical storms as well as for curing VTs.[5–12]

Recent advances in the electroanatomic mapping system (CARTO, Biosense Webster, Diamond Bar, California, USA) has enabled us to create substrate maps of the cardiac chambers, to precisely identify margins of scar, return precisely to areas of interest, visualize lines being created, and perform ablation during sinus rhythm in unstable patients with VT.[5–11] Substrate-guided catheter ablation has become a useful alternative to more conventional ablation methods.[5–11] However, its safety and success rate is not sufficiently high.[5–11]

Recently, RF ablation targeting the low-voltage areas detected by strict voltage criteria guided ablation (SV-substrate-map-ablation) has emerged as an efficient and effective treatment for curing post-infarct VT.[13] However, it is unclear whether or not SV-substrate-map ablation is similarly effective and safe in VT patients with non-ischemic heart disease or whether this method is also efficient and effective for preventing frequent ICD shocks. To clarify this, we continued the VT ablation by using our original method.

The purpose of this study was to address the points identified above. In this article, a search of the medical literature on substrate-mapping guided ablation for electrical storms was also conducted, and the efficacy and safety of the SV-substrate-mapping guided ablation were compared with those of the conventional substrate-guided ablation method in patients with an electrical storm.

Methods

Study Population

From November 2004 to February 2008, 15 consecutive patients (13 men and 2 women; mean age, 64 ± 9 years) that underwent SV-substrate-mapping guided ablation for electrical storms were included after a detailed review of the clinical records of 167 patients who received transvenous ICDs for malignant ventricular arrhythmias. The mean left ventricular ejection fraction (LVEF) was $35 \pm 15\%$ (range, 15 to 58). Eight patients (53%) had a previous myocardial infarction, 5 (33%) idiopathic dilated cardiomyopathy, 1 (7%) cardiac sarcoidosis, and the remaining patient (7%) valvular heart disease (**Tables 1 and 2**). There was no evidence of ischemia, cardiac failure, drug intoxication, or an electrolyte imbalance.

All patients had a history of frequent and recurrent episodes of VT attacks (6.0 ± 3.8/day; range, 3 to 15/day) (**Table 2**) and were in a state of electrical storm at the time of the ablation procedure, which was defined as three or more episodes of VT during a 24 hour period that resulted in a proper ICD shock. In all patients, the clinical VT was documented by a 12-lead ECG at least once; 11 patients (73%) had only one clinical VT, 3 (20%) had 2 VTs, and the remaining 1 (7%) had 3 VTs (**Table 1**). All patients had been treated with antiarrhythmic drugs for ventricular arrhythmias (**Table 1**): Fourteen patients (93%) received a β-blocker, 12 (80%) amiodarone, and 2 (13%) sotalol. The cardiac function was evaluated by echocardiography before and after the ablation procedure.

ICD systems

All patients received a transvenous lead system via the subclavian vein and a pulse generator in the left pectoral region (n = 12, Medtronic Inc., Minneapolis, MN, USA; n = 3, St. Jude Medical Inc., Sunnyvale, CA, USA). A dual chamber transvenous ICD system was used in all patients.

Endocardial Mapping and Ablation

After informed written consent was obtained, electrophysiological testing and catheter ablation were performed based on our recently reported strategy.[13] Three quadripolar electrical catheters were introduced percutaneously via the femoral vein and positioned under fluoroscopic guidance in the high right atrium, His-bundle region, and RV apex. After the catheters were positioned in the heart, 5,000 U of heparin were administered intravenously. The activated clotting time was kept at 250–300 sec during the procedure. A 7 Fr Navistar catheter (Biosense Webstar) with a 4-mm distal tip electrode and 2-mm ring electrodes with 1-mm interelectrode spacing was used. The bipolar signals were filtered between 30 to 400 Hz and displayed at a sweep speed of 100 mm/s on the CARTO system. The fill threshold was set at 10 mm. The peak-to-peak signal amplitude of the bipolar electrogram was measured automatically and confirmed manually. We defined a non-arrhythmogenic area as that having an amplitude of >0.6 mV, low-voltage area as that of >0.1 to ≤ 0.6 mV, and scar area as that of ≤ 0.1 mV without capture by local bipolar stimulation at the maximum output (10 V and 2-ms pulse width) (**Table 3 and**

Table 1 Patient characteristics and all ventricular tachycardias.

No	Age	Gender	Etiology	LVEF (%)	No. of mapping points	Antiarrhythmic drugs	Clinical VT		Inducible VT (Non-clinical)	
							Morphology	CL	Morphology	CL
1	76	M	MI (ant)	40	327	Amio	RBRA	370	RBRA	450
2	57	M	DCM	25	266	Amio + β-blocker	QSLA, QSLA, RBLA	600, 390, 300		
3	50	F	Sarcoidosis	51	362	Amio + β-blocker	RBRA	580	LBRA	600
4	57	M	DCM	58	337	β-blocker	RBLA	290		
5	45	M	MI (ant)	53	281	Amio + β-blocker	RBLA	430		
6	67	M	DCM	18	322	Amio + β-blocker	RBRA	950		
7	65	M	MI (ant)	30	313	Amio + β-blocker	RBLA	550		
8	66	M	MI (inf)	15	366	Sotalol + β-blocker	RBLA	590	RBLA	300
9	57	M	MI (inf)	38	421	Amio + β-blocker	RBLA	300	LBLA	280
10	71	M	DCM	18	272	Amio + β-blocker	LBRA, LBLA	350, 280		
11	69	M	MI (ant)	17	85	Amio + β-blocker	LBLA, RBLA	300, 260		
12	71	M	DCM	29	175	Amio + β-blocker	LBNA	400		
13	65	M	MI (ant)	29	332	Sotalol + β-blocker	RBLA	400	RBRA, LBLA,	330
14	73	F	VHD	55	172	Amio + β-blocker	RBLA	400	QSLA, RBLA	330, 330
15	74	M	MI (ant)	43	177	Amio + β-blocker	RBNA, RBNA	550, 300	LBLA	300
Mean	64	M-13		35	281			430		358
SD	9	F-2		15	95			167		103

The values are the mean ± SD.
Amio: amiodarone, CL: cycle length, DCM: dilated cardiomyopathy, F: female, LA: left axis, LB: left bundle, LVEF: left ventricular ejection fraction, M: male, MI: myocardial infarction, NA: normal axis, No.: number, NW: north west axis, QS: concordant QS pattern, RA: right axis, RB: right bundle, VHD: valvular heart disease, VT: ventricular tachycardia

Table 2 Baseline characteristics of the 160 patients who underwent substrate-mapping guided ablation for frequent ICD shocks.

Authors	No. of Patients	Age, years	Etiology	LVEF, %	Use of class III AADs, %		No. of ICD shocks before ablation
					Pre	Post	
Sra J et al.[5]	19	70 ± 7	Ischemic	27 ± 8	100%	47%	4-62/3 months
Soejima K et al.[6]	40	67 ± 11	Ischemic	29 ± 10	53%	63%	11.9 ± 11.1/2 months
Soejima K et al.*[7]	20	54 ± 14	Non-ischemic	30 ± 11	68%	43%	2-100/month
Schreieck J et al.[8]	5	61 ± 17	Ischemic	18 ± 7	100%	80%	3-310/2 weeks
Volkmer M et al.[9]	13	65 ± 9	Ischemic	30 ± 8	88%	52%	1-930/6 months
Carbucicchio C et al.[10]	48	64 ± 13	Both	36 ± 11	99%	88%	14 ± 8/day
Our cases	15	64 ± 9	Both	35 ± 15	93%	93%	6.0 ± 3.8/days

Values are mean ± SD. *Epicardial substrate-mapping was performed in several patients.
AAD: anti-arrhythmic drug, ICD: implantable cardioverter defibrillator, LVEF: left ventricular ejection fraction, No.: number

Figure 1).[13] All patients underwent SV-substrate-mapping during sinus rhythm with a CARTO system (Biosense Webstar).

During sinus rhythm, a substrate map was carefully created for identifying all, even tiny, low-voltage areas. Arrhythmogenic regions could be considered to be located between scar-scar, scar-tricuspid/mitral annulus, and/or scar-non-arrhythmogenic areas.[13] An arrhythmogenic region of the clinical VT was defined as excellent pace map sites with various stimulus-QRS intervals. If we found a site where the paced QRS morphology was similar to the clinical VT morphology, detailed pace mapping around the site was undertaken to detect as much of the entire arrhythmogenic region as possible. To abolish not only the arrhythmogenic region of the clinical VT detected by pace mapping, but also all possible arrhythmogenic regions within the low voltage areas, a linear ablation design was created according to the following 3 guiding principles:[13]

Table 3 Electrophysiological data and the results and long-term outcome of substrate-mapping guided ablation in 160 patients with frequent ICD shocks.

Authors	No. of target VTs	VT-CL, ms	Criteria of LVAs	No. of RF applications	Procedure time, min	Long-term success rate*	Complication		Follow-up duration (days)
							Death	Others	
Sra J et al.[5)]	2.0 ± 0.7	250–440	<1.0 mV	9 ± 4 lesions	NA	66%	0	1	182 ± 56
Soejima K et al.[6)]	3.6 ± 2.1	263–690	<1.5 mV	18.1 ± 11.2	425 ± 222	47%	0	4	349 ± 286
Soejima K et al.*[7)]	2.9 ± 1.7	215–520	<1.5 mV	17.7 ± 9.4	NA	54%	0	1	334 ± 280
Schreieck J et al.[8)]	5.0 ± 1.9	240–550	<1.5 mV	75 ± 48	340 ± 79	60%	0	0	570 ± 240
Volkmer M et al.[9)]	3.0 ± 2.0	429 ± 82	<1.5 mV	14 ± 6	474 ± 124	54% → 71%#	1	0	750 ± 390
Carbucicchio C et al.[10)]	1–5	381 ± 62	<1.5 mV	NA	260 ± 70	66%	0	8	660 ± 390
Our cases	2.0 ± 0.8	430 ± 167	<0.6 mV	19 ± 13	252 ± 74	67% → 93%#	0	0	801 ± 409

Values are mean ± SD. *Success was defined as freedom from any episodes of ventricular tachycardia. #The success rate improved after the 2nd procedure.

CL: cycle length, LVA: low voltage area, NA: not available, No.: number, RF: radiofrequency, VT: ventricular tachycardia

(1) lesions must cross through the low-voltage area at sites where an excellent pace map is obtained, (2) lesions must extend between scar and scar within the low-voltage area, and (3) lesions must extend from the scar to the non-arrhythmogenic area (0.6 mV). If we could not sufficiently identify the arrhythmogenic region of the clinical VT by pace mapping, targeted pathological myocardium was identified within the low-voltage area by the observation of fragmented or late potentials during sinus rhythm. In those cases, linear RF energy applications were placed along the abnormal potentials.

After the creation of the entire ventricular map, programmed stimulation with up to 3 extrastimuli was first performed from the NaviStar catheter located near the optimum mapping site, then at the RV apex, and finally the RV outflow tract, if necessary, to induce VT. If the induced VT was tolerated hemodynamically, entrainment mapping was performed. An electrically-induced VT was considered the same as a clinical VT when its surface QRS configuration (QRS duration, frontal and horizontal axes, and bundle branch block morphology) was the same as the spontaneous VT and when the difference in the VT cycle length between the induced VT and clinical VT was <40 ms.

As a rule, RF deliveries were not performed in non-arrhythmogenic areas (>0.6 mV). The RF energy was delivered via a 7 Fr 4-mm-tip NaviStar catheter in temperature-controlled mode for 60–90 sec at each ablation site at a maximal target temperature of 55 °C and maximum power of 50 W. The endpoint of the RF delivery was the loss of bipolar pacing capture at a 5-V output and 2-ms pulse width immediately after the RF energy application and by the elimination or significant reduction in the fragmented or late potentials. After the RF delivery at each site, if pacing could capture the ablation site additional RF energy at the same setting was reapplied to the same site for 30–60 sec. If the 4-mm-tip NaviStar catheter could not deliver >20 W of power because of temperature limiting effects, a conventional 8-mm-tip ablation catheter (Ablaze, Japan Lifeline, Tokyo, Japan) was used for energy delivery. An 8-mm-tip catheter was also adopted in patients undergoing a second endocardial procedure. In such cases, because the open-irrigation catheter was not available in Japan at that time, we had to insert 2 catheters simultaneously into the ventricle: the 4-mm-tip NaviStar catheter, which was used only for mapping, and the 8-mm-tip conventional catheter which was inserted from the contralateral femoral vessel and used only for ablation. We positioned both catheters at the same site under guidance with fluoroscopy and the local potentials, and delivered the RF applications with the 8-mm-tip catheter at the target site navigated with the 4-mm-tip NaviStar catheter. The endpoint of the procedure was the non-inducibility of any clinical VT by programmed stimulation with up to 3 extrastimuli.

Epicardial Mapping and Ablation

Epicardial mapping was performed in case 2 using a percutaneous transcardial approach as reported previously.[7,14)] Briefly, the epicardial ablation utilized a subxiphoid approach with an epidural needle using a 10 ml syringe with contrast media, the needle was advanced toward the left scapula guided by fluoroscopy. When the pericardial space was thought to be reached, a small bolus of ≤1 ml of contrast agent was injected. A 7 Fr sheath used for standard ablation was advanced over the guidewire which was introduced into the pericardial space. A 4-mm-tip NaviStar catheter was introduced through the sheath

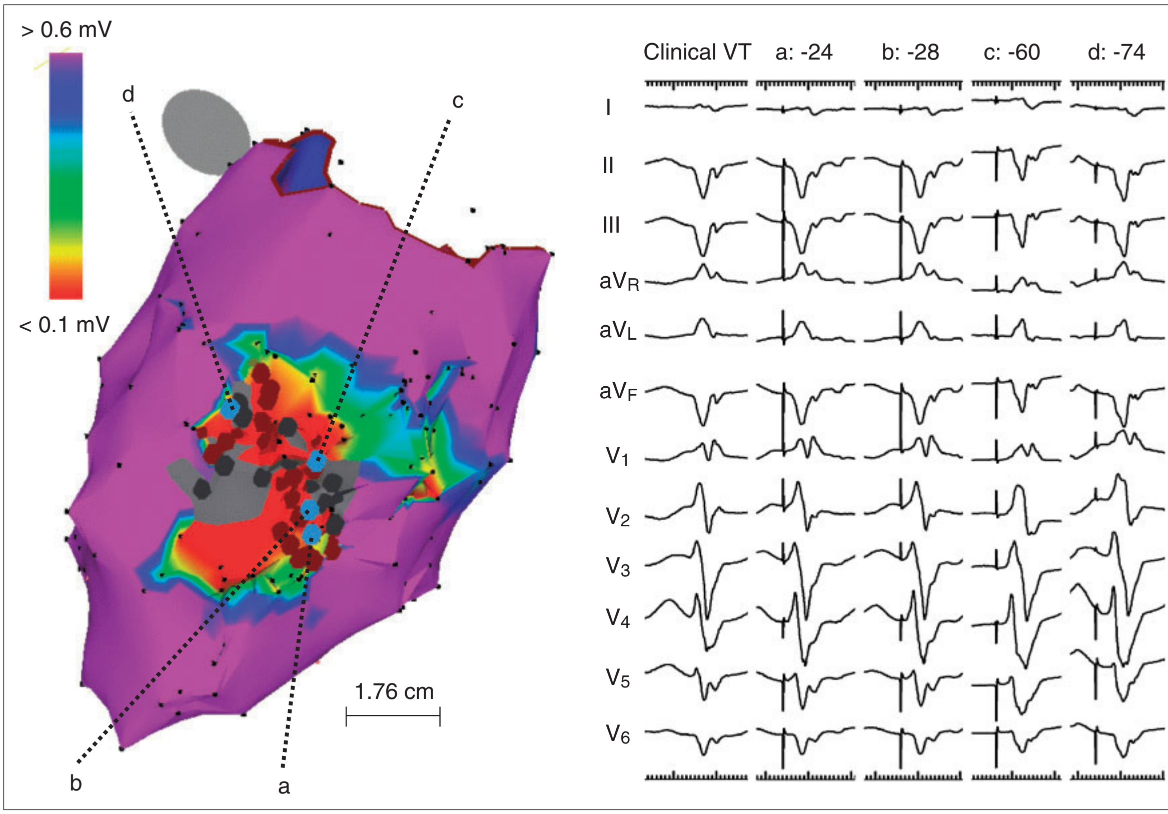

Figure 1 Representative case of strict voltage-based substrate-mapping in a patient with an inferior infarction (case 9). Elaborate original voltage criteria were used to define the non-arrhythmogenic area as >0.6 mV (purple); low-voltage area as >0.1 to ≤0.6 mV (blue to red); and scar as ≤0.1 mV (gray). The brown and blue tags represent the ablation site and optimum pace map sites, respectively (a-d). The 12-lead electrogram exhibits the clinical ventricular tachycardia (VT) and pace maps at sites a-d. The number following each letter indicates the stimulus-QRS intervals; the QRS morphology at all sites is identical to the clinical VT morphology. The "a" site is considered to be the exit of the VT reentrant circuit.

into the pericardial sac. The exposed epicardial surface was mapped during sinus rhythm using fluoroscopy and an electroanatomic mapping system with our original elaborate voltage criteria. The RF deliveries were similarly performed from the endocardial approach, as mentioned previously.

Follow-up

The patients remained hospitalized under continuous rhythm monitoring for 1–2 weeks after the ablation procedure. After leaving the hospital, the patients underwent follow-up at 2 weeks post-procedure, and then every 1 to 2 months thereafter at our cardiology clinic. Any delivered ICD therapies were noted. When interrogation of the ICD allowed determination of the treated VT cycle length, the treated VT was considered consistent with the ablated VT if the cycle length of the treated VT was within 20 ms of the ablated VT cycle length.

Literature search and data collection

Using the PubMed database (MEDLINE, National Library of Medicine), we conducted a comprehensive search of the literature encompassing the period from 1998 to 2008. We searched for the key words "ventricular tachycardia," "substrate," "mapping," "ICD", "electrical storm" and/or "catheter ablation". We then reviewed the articles identified by our search using prospectively defined inclusion criteria. The articles were considered for inclusion if they were published in the English language and described at least five cases of substrate-mapping guided ablation for an electrical storm. From the obtained data, we examined the clinical, ECG, and electrophysiological characteristics of the electrical storm, and compared our results of SV-substrate-mapping guided ablation with those of previous studies (**Tables 2 and 3**).

Statistical analysis

The continuous variables are expressed as the mean ± SD, and were compared using the Student's *t* test. A p value of <0.05 was considered significant.

Results

Electrophysiological study, VT characteristics and results of the RFCA

The VT originated from the LV in 13 patients (87%), and the RV in the remaining 2 (13%). With programmed ventricular stimulation, the mean number of induced monomorphic VTs was 2.0 ± 0.8 (range, 1 to 3), and mean cycle length of the clinical VTs 430 ± 167 ms (**Tables 1 and 3**). Clinical VTs were inducible in 13 patients (87%) during the ablation procedure. Entrainment mapping during the VT could be obtained only in 5 patients (33%) because of hemodynamic instability. The number of optimal pace map points was 4.9 ± 2.0 points/patient (range 3–8). The number of mapping sites was 281 ± 95 points/patient (range 85–421) (**Table 1**).

In the first ablation procedure with the endocardial approach, the clinical VTs were successfully eliminated in 10 patients (67%) (**Table 3, Figures 2A and 2B**). In the remaining 5 patients (33%) who suffered a recurrence of VT within a week after the first procedure, a second ablation session was performed within a week from the first procedure. In 4 patients (27%) with recurrent VT, the targeted VTs were controlled with a second endocardial procedure. However, no targeted areas were found during mapping from the endocardial sites of both ventricles in the remaining patient. Therefore, an epicardial approach was performed, and the clinical VTs were successfully ablated in the second procedure (**Figure 2A**). Finally, in all patients, the frequent ICD deliveries were completely controlled after the second procedure (**Figure 2B**).

During the electrophysiological testing, 9 non-clinical VTs that had a different QRS morphology than the clinical VTs were also induced in 7 patients (47%) (**Table 1**). In 4 patients (27%), no types of VT were ever induced during or after the procedure (**Figure 2A**). The total duration of the ablation procedures was 252 ± 74 min. The total number and duration of the RF energy applications were 19 ± 13 and 23 ± 17 min, respectively (**Table 3**).

Follow-up

The LVEF and left ventricular end-diastolic dimension (LVEDD) measured by echocardiography 1 week after the ablation procedure were 36 ± 14% and 61 ± 12 mm, respectively, both of which were not significantly different from those measured before the procedure (LVEF, 35 ± 15%; LVEDD, 61 ± 12 mm).

During a mean follow-up period of 801 ± 409 days (median 677 days; range, 397–1583), VT and appropriate ICD shocks were observed in only 1 patient (7%) (**Figure 3**). No complications were observed during or after the RFCA procedure and no inappropriate ICD shocks occurred during the follow-up period. No patients died during the follow-up period (**Table 3**).

Literature review

The selected features from 145 cases of substrate-mapping guided ablation for frequent ICD shocks and from the present 15 cases are summarized in **Tables 2 and 3**.[5–10] Although all previous reports and our present study performed substrate mapping with the CARTO system, the detailed mapping strategy, settings of the RF delivery, and ablation catheters differed with each article.[5–10] There were substantially more males (89%) than females (11%). A majority (78%) of patients had a previous myocardial infarction, and the mean LVEF was 30 ± 6%. All had been given ICDs for ventricular arrhythmias and suffered from frequent appropriate ICD shocks. The baseline characteristics, including the prevalence of the use of antiarrhythmic drugs and frequency of appropriate ICD shocks in our cases were compatible with those in the previous studies (**Table 2**).

The electrophysiological data, results and long-term outcomes of substrate-mapping guided ablation are shown in **Table 3**. In all studies except for our study, the low-voltage area was defined during sinus rhythm by electrograms with an amplitude of <1.0 or 1.5 mV; dense scar was defined by electrograms with an amplitude of <0.5 mV. SV-substrate-mapping was only adopted in our study. The overall success rates after conventional substrate-mapping guided ablation was 47% to 67% after the first procedure, and potential complications occurred in some patients: 1 patient (0.6%) died in the hospital after the procedure,[9] and other procedure-related complications were reported in 14 cases (8.8%).[5–7,10] Compared with the results of the conventional substrate-mapping guided ablation, the total procedure duration was shorter in our results of SV-substrate-mapping guided ablation. The long-term success rate of our study increased up to 93% after a repeat ablation procedure in 5 patients, which was higher than that of the previous studies.

Discussion

Main findings

The results of the present study demonstrated the

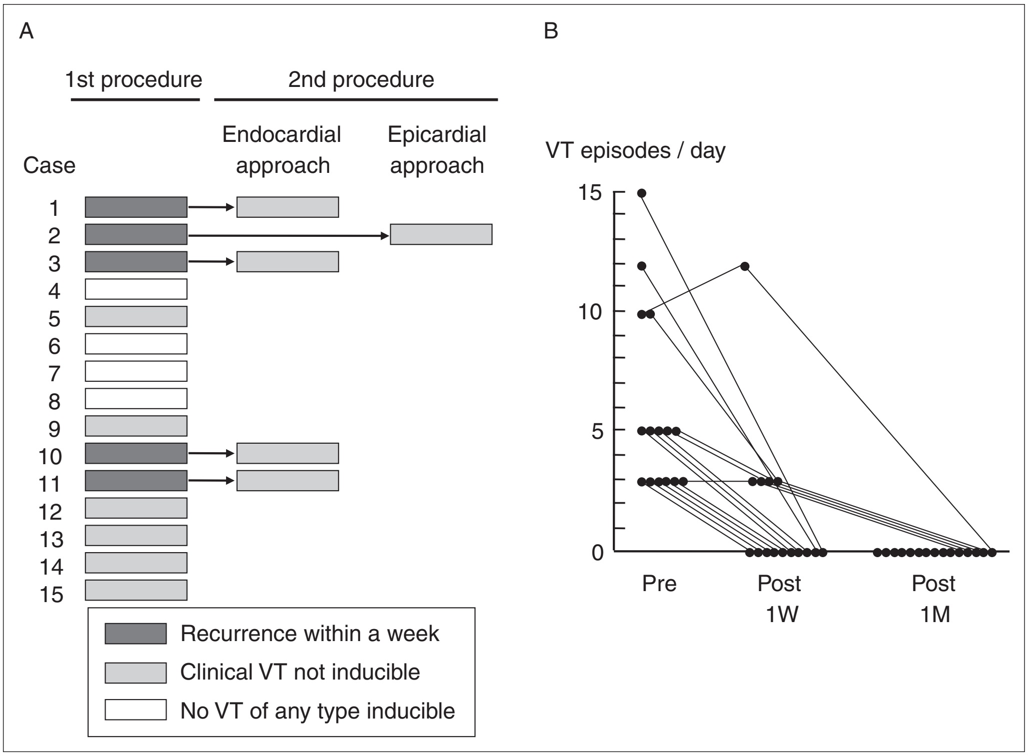

Figure 2
A. Immediate ablation results.
Electrical storms in 10 patients (67%) were successfully controlled in the first procedure. Four patients (27%) underwent a second endocardial catheter ablation. An epicardial procedure was performed in 1 patient.
B. Acute ablation results.
The frequency of the VT episodes is shown for the period before (pre) and the following intervals: one week after the ablation and from one week to one month after the ablation. M: month, VT: ventricular tachycardia, W: week

following findings: 1) In both the ischemic and non-ischemic patients the SV-substrate-mapping guided ablation abolished the clinical VT, then resolved the electrical storm completely with no potential complications; however, five of them required a second ablation session; 2) During a moderate follow-up period of >2 years, only one VT followed by an appropriate ICD shock was observed; 3) Our results and data of the ablation procedure and follow-up, including the procedure and RF energy application times, were comparable to or better than those of the previous studies of substrate-mapping guided ablation.

These findings indicate that the SV-substrate-mapping guided ablation may be effective and safe for resolving serious patient situations and to prolong the longevity of the ICD device in patients with electrical storms, irrespective of the underlying heart disease.

Clinical importance of reducing ICD therapies

A significant decrease in ICD therapy deliveries may be clinically important, not only for quality-of-life, but also the reduction in subsequent adverse cardiovascular events. Previous trials found an increase in congestive heart failure and mortality in patients receiving ICD shocks.[1,2] Furthermore, both appropriate and inappropriate ICD shocks are associated with a marked increase in the subsequent risk of cardiac death in patients with heart failure.[15] The negative inotropic consequences of multiple shocks themselves could increase the risk of death.[16,17] These results imply the need for a more aggressive application of catheter ablation for patients suffering from frequent shock deliveries.

In the present study, we could suppress the electrical storms completely with catheter ablation in all patients. During the long-term follow-up period after the ablation, only one appropriate ICD shock and no inappropriate ICD shocks were observed. Furthermore, no patients died during the follow-up period. Therefore, in our study, there was a high possibility that the significant reduction or abolishment of the VT attacks and the resulting defibrillator shocks by catheter ablation therapy might improve the subsequent prognosis.

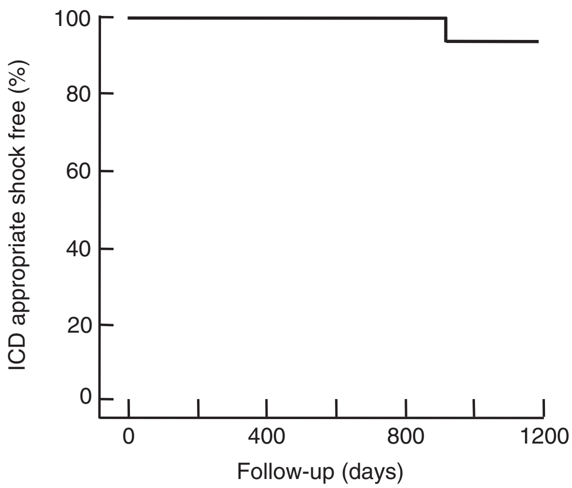

Figure 3 Long-term ablation results.
Kaplan-Meier curve demonstrating an event-free rate of 93% during the follow up period. Only one sustained ventricular arrhythmia followed by appropriate shock was observed in case 13.

Efficacy and safety of strict voltage-based substrate-mapping from an endocardial approach

The usefulness of ablating drug-refractory VT was clearly demonstrated previously; however, the long-term success rates did not reach sufficiently acceptable results.[5–10] In the present study, the SV-substrate-mapping guided ablation procedure had a sufficiently high acute success rate and better long-term outcome compared to that of substrate-mapping guided ablation with conventional voltage criteria. The total procedure time in this study was similar to previous catheter procedures, most of which was spent on SV-substrate-mapping in this study. A recent study showed that, using the SV-substrate-mapping, the targeted arrhythmogenic area ($\leq$0.6 mV) for the RF energy delivery was restricted approximately to half the size compared to that of the conventional targeted area with an amplitude of $\leq$1.5 mV.[13] Therefore, in SV-substrate-mapping, the abnormal regions could be identified simply and clearly during the procedure by restricting the target regions within smaller arrhythmogenic areas, which may result in a shorter procedure time compared to the previous results.

Several ablation-related complications, including anticipated complete atrioventricular block, transient ischemic attacks, and pericardial effusions, have been reported in 14 patients (8.8%) who underwent substrate-mapping guided ablation.[5–7,10] Unfortunately, 1 patient (0.6%) died in the hospital the night after the procedure from electromechanical dissociation after cardiac tamponade (**Table 3**).[9] On the other hand, neither procedure-related deaths nor cardiac complications were observed in the recent studies of SV-substrate-mapping guided ablation, including this study.[13] In this study, SV-substrate-mapping was also useful for determining the optimal ablation sites in the patients with nonischemic heart disease as well as those with ischemic heart disease. Because the target sites of the RF energy applications determined by SV-substrate-mapping were always smaller than those determined by the conventional voltage criteria, we think that the futile applications of RF energy at the myocardium within or near the low-voltage areas, which were not related to the VT, could be avoided. Fewer applications of RF energy might result in less injury to the myocardium and avoidance of deterioration of LV function after the ablation procedure. Further, fewer applications of RF energy and a shorter procedure duration may also contribute to a reduction in the complications, such as heart failure or thromboembolic events, and result in a favorable long-term outcome.

Strict voltage based substrate-mapping from an epicardial approach

Recent studies demonstrated that the patients with nonischemic cardiomyopathy sometimes had no scar tissue in the LV endocardium, but exhibited a profoundly large low-amplitude region in the LV epicardium,[18] and that reentrant VT associated with nonischemic cardiomyopathy was successfully eliminated in only 54% of patients from the endocardial approach.[7] Therefore, epicardial mapping should be considered when no suitable ablation sites exist within the LV endocardium in patients with nonischemic heart disease. Indeed, in our case with idiopathic dilated cardiomyopathy (case 2), no appreciable low-voltage areas were visualized in the LV endocardium and therefore no ablation lesions were placed in the first procedure. The clinical VTs were eliminated and the electrical storm was completely suppressed only by a catheter ablation from an epicardial approach. Low-amplitude regions were found from the LV anterior wall to the apex. The targeted arrhythmogenic area ($\leq$0.6 mV) for the RF energy deliveries was restricted approximately to half the size of the generally targeted area with an amplitude of $\leq$1.5 mV in this case. The abnormal regions were easily delineated and made lucid during the procedure only because SV-substrate-mapping was employed. Therefore, we think that SV-substrate-mapping may also be useful for curing VT and avoiding complications compared to the risks inherent with the futile and numerous applications of RF energy with the epicardial approach.[14]

Study limitations

First, the patients were systematically maintained on the same oral antiarrhythmic regimen as before the ablation. In particular, a majority of patients were treated with oral amiodarone after the procedure. These drugs probably contributed to the slowing of the VT and may have rendered the VT more amenable to mapping and ablation. Without the concomitant use of antiarrhythmic drugs, our immediate success rate and long-term outcome may have been lower. Actually, in case 13, drug-induced bradycardia later developed, so sotalol and the β-blockers were discontinued. Unfortunately VT and appropriate ICD shocks were observed two weeks after the reduction of those drugs. Hence, we can assume that ablation and drug therapy will remain important components in managing patients with frequent ICD therapies.

Second, the previous studies reported that the VT isthmus resided not only in dense scar (<0.5 mV), but also in the border zone (>0.5 to <1.5 mV).[19,20] On the other hand, Hsia et al. demonstrated that 47/56 (84%) of VT entrance or isthmus sites were located in <0.5 mV regions.[21] In agreement with the previous results,[13,21] we reached a sufficiently high success rate without any RF energy applications to preserved voltage areas (>0.6 mV). However, the reason for the difference between the previous results[5–10,19,20] and SV-substrate-mapping guided procedure is still unclear.

Third, we created a linear ablation lesion to abolish not only the arrhythmogenic region of the clinical VT detected by pace mapping but also all possible arrhythmogenic regions. Moreover, if we could not completely identify the arrhythmogenic region of the clinical VT by pace mapping, RF energy was delivered to putative pathological regions with fragmented or late potentials. One of the endpoints of the RF deliveries was the elimination or significant reduction in abnormal potentials. Additional RF deliveries at those sites may have improved the clinical outcome in the present study.

Finally, the number of patients was not large enough, so a large-scale completely prospective study may be needed to further assess the prognostic value of SV-substrate-mapping guided ablation compared to conventional substrate-mapping guided ablation.

Conclusions

Electrical storms were completely suppressed in all non-ischemic patients as well as ischemic patients by SV-substrate-mapping guided catheter ablation. The clinical VTs were successfully eliminated and the clinical outcome was also sufficiently good. Additionally, no procedure-related deaths or any complications were observed during the long-term follow-up period. Our elaborate voltage criteria guided catheter ablation may be one of the ideal strategies for patients with frequent ICD shocks.

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